e.—GENERAL PATHOLOGY OF THE NERVOUS SYSTEM.

EPILEPTIFORM CONVULSIONS FROM CEREBRAL DISEASE.—J. Hughlings Jackson. The simplest convulsions are those described by Bravais in 1824. They are here spoken of as epileptiform seizures to distinguish them from epilepsy proper. The study of these seizures has entered into the scientific stage principally consequent on Hitzig and Ferrier's physiological researches, and the clinical researches of Charcot and other physicians. The seizures alluded to begin in the arm or face, or leg; each is believed to depend on an excessive liberation of energy (excessive nervous discharge) by cells of some particular part of the cortex, within the so-called motor region of the cerebrum.

1. *Starting-points.*—In all cases the spasms begin unilaterally. *a.* Hand, usually index finger, or thumb, or both. *b.* Face, usually near mouth, or in the tongue, or in both. *c.* Foot, usually great toe.

2. *Ranges* are very numerous. We may arbitrarily make three ranges. *a.* Mono-spasm (arm, face, leg; clinically analogous to Charcot's monoplegias from cortical destructive lesion). *b.* Hemi-spasm. 1. Of the face, arm, and leg, with turning of the head to the side convulsed, clinically analogous to a degree of cerebral hemiplegia, described by Vulpian and Prevost, in which the head and eyes turn from the side paralyzed. In this range there is usually some spasm of the bilaterally acting muscles of both sides. Part of the evidence verifying Broadbent's well-known hypothesis as to differences in the representation of the unilaterally acting muscles in the two sides of the cerebrum. *c.* The other side of the body or part of it may be convulsed. This has been explained variously. 1. On the facts of Wallerian wasting "descending" into the same side (anterior column), and into the opposite side (lateral column), of the cord. 2. By Broadbent's hypothesis. 3. By the opposite cerebral hemisphere becoming engaged.

3. *March of spasm.*—When limited to an arm, the spasm may go down that limb; but usually it goes up. If in hemi-spasm the spasm begins in the hand, it goes up to the arm and down to the leg; if it begins in the foot it goes up the leg, and, with many exceptions, down the arm.

There are few observations on the spreading of spasm (when the convulsion has become bilateral) on the opposite side. Questions as to spreading of spasm on "opposite," in relation to starting-point on first side.

We have further to note that the spasm spreads in compound order: this is most easily seen in the face. First, spasm of muscles whose movements are largely unilateral in action on one side of the face. Next there is a compound effect of greater spasm of them, with added spasm of the bilaterally acting muscles of both sides. (Further evidence of Broadbent's hypothesis.)

4. *Suddenness of onset, rapidity of spreading, and duration of seizures.*—The author thinks that the more suddenly the spasm starts and the more rapidly it begins to spread, the greater the range and the shorter the seizure (*vice versa* for deliberately, etc., beginning fits). Inference that the cortical liberation of energy in different cases varies not only in quantity liberated, but in rate of its liberation—the more rapid liberations overcoming greater resistances.

5. *Post-paroxysmal condition* (*paralysis*). — Admitting many complications and difficulties, ordinary temporary paralysis after convulsion, beginning in a patient who before a seizure had no obvious paralysis, is spoken of. The paralysis is, in the author's experience, always of the parts first and most convulsed. It varies in degree from such as mere inability to pick up a pin to absolute powerlessness of the hand and arm. It varies greatly in range, from paralysis of the hand to hemiplegia, with (J. Mickle) lateral deviation of the eyes, and probably to greater ranges still. There is, for an example, this sequence: *a.* A man is seemingly well. *b.* His leg is convulsed strongly and the arm slightly for about ten minutes. *c.* The leg is much paralyzed for a few hours. *d.* He is seemingly well again. When a cortical lesion is found we have to note whether any paralysis there had been was after a convulsion or not.

Various hypotheses as to the nature of post-epileptiform paralysis: *a.* Congestion induced by asphyxia in the paroxysm. But there is no asphyxia in some local convulsions leaving complete local paralysis. *b.* Extravasation of blood. There is in the cases just adverted to nothing to cause extravasation. *c.* Todd and Alexander Robertson suppose that after the excessively active processes from cortex to muscles which the convulsion implies, there is corresponding exhaustion of the parts engaged, or of some of them—internal capsule, fibres of the cord, anterior horns, nerves, muscles. This hypothesis, the author thinks, is most in accord with the facts. The objection that there is no relation of proportionality between the severity of the seizure and the subsequent paralysis, is considered.

The author refers to conditions of the deep "reflexes" after such seizures. He first alludes to Westphal's observations on them after epileptic seizures, and to some more recently made on such cases by Dr. Beevor. He points out the difficulties as to the state of the reflexes in post-epileptiform paralysis, and hence the as-yet doubtful bearing of his observation on the various hypotheses stated. The state of the "reflexes" should be noted instantly after the paroxysm, and at various times up to recovery. If the "reflexes" are exaggerated on the side temporarily paralyzed, there is the difficulty that they may be more or less so permanently from permanent changes induced in the cord by *destructive* disease of the cortex; for exaggerated knee-jerk and foot-clonus may be found on the side subject to convulsion several weeks after a fit, even when there is no obvious paralysis of the leg.

6. *Post-epileptiform aphasia.*—Partial temporary aphasia is found in some cases after fits beginning in the right cheek; often, however, after there is what is called ataxy of articulation. After fits beginning in the hand the partial temporary aphasia is often a misuse of words, or a reduction to well-organized propositions, such as "I don't know," "very well." There may be some temporary right-sided paralysis along with the temporary aphasia. Inability to put out the tongue when told may exist in these, as in other cases of aphasia, when that organ moves well in all automatic operations. Todd and Robertson's hypothesis, the author thinks, explains best the temporary aphasia (the negative element only, of course). Further, he thinks that it best accounts for the negative physical condition during loss of consciousness after seizures of epilepsy proper, whether there be the positive condition of mania, or other actions as well, for which positive condition it indirectly accounts ("loss of control," Anstie, Dickson).

7. *On affection of consciousness.*—Usually unaffected in limited convulsion of a limb, side of face, or even of one side of the body. Roughly speaking, consciousness usually ceases when the eyes and head begin to turn to the side first convulsed. The more sudden and rapid the spasm, the less is the range attained before consciousness is lost. The severest epileptiform seizures differ from the severe seizures of epilepsy proper as to affection of consciousness, in that, in the former, consciousness ceases late in the paroxysm, in the latter, the first thing, or very early.

8. *Locality of lesion* (anatomical diagnosis).—When disease is discovered *post-mortem* to the epileptiform seizure it is usually in the so-called motor region. Cases referred to.

9. *Physiology of lesion.*—When the fits always of the same style recur, although often in different degrees or ranges, the inference is that there is a persistence of change in some cells in one locality, such that they attain high instability, and occasionally discharge excessively.

10. *Pathology of the lesion.*—The question in pathology is "How does the hyperphysiological condition of instability result?" In some cases the author has not discovered, has no doubt overlooked, a local lesion; in many cases there is tumor. Clinically there is in some cases a condition for embolism; in some there is disease of one ear, evidenced by discharge, and of the side opposite that on which the convulsion begins. In some cases the seizure sets in, in same part on the side opposite to the side of the head injured—there being in a few of these cases a local depression of the skull.

11. *Treatment.*—Partially empirical (bromide, etc.), ligature, and treatment of syphilis. Question of trephining in certain cases.—*Abstr. Internat. Congr.*, 1881, p. 76.

Pathology of Basal Brain Tumor, with Demonstration of a very Rare Case.—Dr. F. Müller, Graz. Conclusions: 1. Basal interruption of conductivity of the trigeminus, as well as destruction of the Gasserian ganglion, are not necessarily followed by morbid changes in the nutrition of the eye. 2. Absolute paralysis of the first branch of the trigeminus may exist—at least for many weeks, without trophic changes on the part of the eye being produced, notwithstanding the want of any artificial means of protection. 3. In spite of basal interruption of the conductivity of the facialis, the Faradic excitability of the neuro-muscular apparatus may remain normal for some time—the particular case for four weeks (at the most there occurs in some branches a very small quantitative diminution of excitability). The non-existence of reaction of degeneration during the first four weeks of facial paralysis proves nothing as to the formation of a basal tumor. 4. In complete paralysis of all the oculo-motor branches which innervate the outer muscles of the eyeballs, and in simultaneous complete amaurosis from purely basal causes, there may exist normal pupillary reflex to light and shade. 5. This particular case showed the simultaneous existence of *eleven alternating forms of paralysis*, among them an alternating sensory paralysis of the trigeminus and of the extremities, inclusive of trunk. 6. The

motor hemiplegia entailed by the destruction of one half of the pons is persistent, while the sensory paralysis is only of a *transitory* nature.

7. *One* intact half of the pons is sufficient for the conduction of the entire sensibility of all the extremities, and of the trunk.

8. Destruction of the middle crus cerebelli produced in my case involuntary *manége* movements, and a falling toward the opposite side.—*Abstr. International Med. Congress*, 1881, p. 89.

Localization of Disease in the Brain and Spinal Cord so far as Pathognomonic and Diagnostic.—C. E. Brown-Séquard. The author states in a preliminary paper: "Under this title it is my purpose to ask the Section to discuss the following questions: 1. Are there parts of the brain and of the spinal cord which, being diseased, give rise to symptoms which no other parts can produce? 2. What is the diagnostic value of certain symptoms to show the seat of disease in the brain or spinal cord? 3. What gains have we made in recent diagnosis by the recent researches on localization of disease in the cerebro-spinal centre? As regards the first of these questions, I will try to show that although there is no symptom which alone possesses an absolute pathognomonic value concerning the seat of the disease, there are, however, morbid manifestations, the co-existence of which, establish almost certainly, and sometimes even certainly, that special parts are diseased. As regards the second question, it will lead me to speak of the connection: 1st, of aphasia with disease of the third frontal convolution, the island of Reil, and the occipital lobe on the left or on the right side; 2d, of the Jacksonian convulsions with some cerebral convolutions; 3d, of brachial, crural, facial paralysis and of other kinds of monoplegia with lesions of certain convolutions; 4th, of cerebral semi-anæsthesia with disease of the optic thalamus or of the posterior part of the internal capsule; 5th, of hemi-chorea with disease of the corpus striatum or of the anterior part of the internal capsule; 6th, of titubation with disease of the cerebellum and of some parts of the bone of the brain; 7th, of diabetes with disease of the floor of the fourth ventricle; 8th, of labio-glosso-laryngeal paralysis with certain groups of bulbar nerve cell; 9th, of some symptoms of labio-locomotor ataxia with disease of certain parts, and of other of the symptoms of that affection with disease of other parts, of the posterior columns of the spinal cord; 10th, of paranæs-

thesia with disease of the central parts of the lumbo-dorsal enlargement of the spinal cord; 11th, of progressive muscular atrophy with atrophy of the nerve cells of the anterior gray cornua of the spinal cord; 12th, of the essential infantile paralyses with small *foci* of inflammation of the part of the gray matter just named; 13th, of intermittent paraplegia with ischæmia of the dorso-lumbar enlargements of the spinal cord. As regards the third question, I shall show that we have recently made considerable advances, although much less than is generally believed.—*Abstr. International Med. Congress*, 1881, p. 71.

On Percussion of the Skull in the Diagnosis of the Brain.—Dr. Alex. Robertson, Glasgow. Although attention was directed by the writer in 1877 to the value of percussion of the skull in the localization of disease on the surface of the brain, and Dr. Ferrier (*Brain*, 1879) has also insisted on its importance, the subject may still be considered comparatively new to the profession. The paper first deals with objections against the practicability of transmitting the degree of force employed in tapping the skull with the finger to the surface of the brain. Duret's experimental researches on cerebral traumatism show that in blows on the head a "cône de dépression" is formed, which passes deeply in the line of the thrust to the base of the skull; the slight force of percussion will act in a similar way, though it may scarcely extend beyond the cortical substance. A somewhat analogous instance of the irritation of a morbid part into conscious sensibility is sometimes supplied by disease of the lungs, in percussion over a cavity or softened caseous matter near the surface of the lung. Clinical experience is one apparently conclusive on the question. Cases of Jacksonian epilepsy and monoplegias are referred to, when the symptoms pointed to the motor regions of the convolutions as the seat of the disease, and in which percussion of the skull gave very distinctly deep-seated pain in that part of the head, and *nowhere else*. The two kinds of symptoms—the disturbance of function and the developed pain—lend each other mutual support in the localization of the disease. When the convulsive movements are general, the pain brought out by percussion at some other part of the head, probably indicates the centre from which the morbid action spreads to the motor convolutions. In some cases corroboration of the diagnosis is derived from the diagnosis of former blows, and also from the beneficial effect of

treatment over the painful region: another objection is, that the brain substance is wholly insensitive, and the membranes are only slightly sensitive. In reply, it is pointed out that the dura mater and the pia mater, like other fibrous membranes, when in a state of disease and subject to tension, may give rise to sever pain. The pain in the cases founded on is not induced by mere rubbing or gentle pressure, but by *percussion*. It is therefore inferred that the disease is not in the bone, unless it be in the inner table of the skull; and if it is situated in this part, it is of great importance to elicit the fact, as morbid action there usually involves the outer, and often the inner, membrane and brain itself. However, disease of the bone in adults in most cases is syphilitic, and the pain, as a rule, is such as to stand in no need of artificial development to manifest its existence. Then follow brief notes of six cases under the writer's care. In some of these great benefit was derived from repeated counter-irritation over the seat of developed pain. It is stated that several cases support the prevailing views respecting the localization of motor function in the cortical substance. The mode of practising percussion of the skull is described. The physician should be careful to make the taps of the finger as nearly as possible of equal force, except in the temporal region where they should be lighter. It is well to percuss one's own head previously, to ascertain the character of the tap which can be borne without discomfort. It is advised to avoid, either by remark or otherwise, directing the patients' attention to any particular part of the head, particularly if they be of an impressionable or hysterical disposition. It is not claimed that this means of diagnosis will be of very wide application. It probably will not be of service if the morbid action be diffused, as in ordinary cases of insanity. It is chiefly of use when the disease is limited in extent, and particularly if it is attended by gross products, such as inflammatory lymph, producing local tension or tumors of the surface, or in the membranes. In injuries of the head, it may occasionally be of service. Thus, in a somewhat doubtful case of fracture of the skull the writer has seen it assist in marking out the line of the fracture. In disease of the inner table of the skull, when the pain of the head is wide-spread, it may help to localize the lesion. Wherever, therefore, there is the least ground, judging from the general symptoms, for suspecting that disease may exist superficially within the skull, percussion of the head should not be omitted; it may yield most valuable information on which important local treatment may be based.—*Abstr. Internat. Med. Congress*, 1881, p. 85.

PERFORATING ULCER OF THE FOOT AS CONNECTED WITH PROGRESSIVE LOCOMOTOR ATAXY.—Prof. Ball, Paris. Rep. by Dr. Thibierge. 1. Perforating ulcer of the foot is in such cases the consequence of the spinal disease, as in "joint disease," which has been brought before the public by Charcot and myself. 2. The local disease is more especially connected with certain symptoms of locomotor ataxy, such as shooting pains, absence of the tendon reflex, and other trophical lesions. 3. The perforating ulcer may be cured while the symptoms of locomotor ataxy follow their progressive course.—*Abstr. Internat. Med. Congress*, 1881, p. 97.

RUMPF ON THE ACTION OF LYMPH ON THE NERVE CENTRES. (*Pflüger's Arch.*, xxvi, p. 415).—Rumpf maintains the correctness of his observations that the axis-cylinder of nerve is dissolved by lymph after passing through a preliminary stage of swelling. The swelling of nerve stump under the influence of lymph is at first of the nerve proper, and only later consists in an inflammatory hypertrophy of the connective tissue. The author was led to examine the analogous event as regards fibres and cells of the brain and cord. The results on frogs are very striking; the skull having been opened, a portion of brain isolated, and the wound reclosed, Rumpf found, forty-eight hours later, that nothing remained of the isolated portion of brain but a small quantity of connective tissue. The spinal cord having been laid bare, transected above and below, and isolated from all nerves, Rumpf found, at the end of twenty-four hours, that all the nerve elements had begun to swell, this being still more marked at the end of forty-eight hours, after which the cord became amorphous and rich in nuclei, and (five or six days) disappeared, leaving nothing but a little connective tissue. This absorption does not ensue upon section of the cord without section of its nerves, nor when the cord and intervertebral nerves having been cut, the cauda equina is left intact. Nor is it produced if, in addition, all sensory roots of the cauda are divided; whereas if its motor roots are alone divided, absorption follows, though not so rapidly or completely. He obtained similar results with hardly any interference with the circulation. These observations indicate a centripetal trophic influence, partly via sensory channels, but chiefly via motor channels, and confirm the views of Kühn and of the author (1860), that from end plate to centre there exists a constant excitatory and trophic influence. The author also experimented

on the brains of pigeons and found, as in the case of frogs, swelling; absorption was, however, usually accompanied by suppuration.

The author concludes that, to the maintenance of centres, their union with efferent channels is necessary, and that, failing this union, the organ degenerates, and is absorbed by action of the lymph; also that the union alone is sufficient to preserve the otherwise isolated cord, and that even partial union suffices by vicarious action to maintain the ventricle of the whole organ. This retrograde influence is the antithesis of tonus, and Rumpf therefore terms it "retrotonus." Thus, as laid down by Sigmund Mayer, the nerve fibre with its two end organs, central and peripheral, constitutes a trophic as well as functional unit (A. Waller, in *Brain*, Jan., 1882, p. 571).

THE PATHOLOGICAL HISTOLOGY OF THE SPINAL CORD.—S. G. Webber (Report, Med. & Surg. Report, of City Hosp., Boston, 3d S., 1881). The author presents a summary of the normal histology of the spinal cord, following which is a consideration of the pathology of the different histological elements, and a description of the pathological histology in different forms of myelitis. A summary of the latter is as follows:

1. *Acute interstitial myelitis*, with swelling of the fibres, nuclei, and cells of the neuroglia, with destruction of nerve fibres and nerve cells, leading to softening.

2. *Acute parenchymatous myelitis*, where the nerve fibres in the white substance are primarily or chiefly affected, myeline and axis-cylinders both disappearing, but the interstitial tissue remaining, seemingly not much changed; also cases in which the nerve cells are chiefly affected, especially those of the anterior cornua, the nuclei and cells of the neuroglia being almost entirely exempt from change, as in infantile paralysis and allied affections.

3. *Chronic interstitial myelitis*, affecting the neuroglia, fibres, nuclei, and cells in both white and gray substance, the nerve fibres and cells being affected only secondarily, as in sclerosis.

4. *Chronic parenchymatous myelitis*—in the white columns only, locomotor ataxia—or lesion of the posterior columns (and secondary ascending and descending degeneration possibly), is as yet well known; lateral sclerosis probably belongs to this variety. In the gray substance the cells are affected as in progressive muscular atrophy. There is as much reason to thus subdivide myelitis as there is to divide nephritis into the interstitial and parenchymatous forms.

CEREBRAL TUMOR INVOLVING THE NERVES OF THE SPECIAL SENSES.—Dr. Lucian Howe, Buffalo. A male, æt. 27, had been troubled with frontal headaches for a year, about which time failure of vision began. On examination, the fundus of the right eye exhibited an illy-defined outline of the optic disc, enlarged and tortuous vessels often lost from sight when traced from their starting-point, being covered by œdematous and swollen retinal tissue. The left eye presented similar changes, though with less involvement of the nerves. Right vision sufficient to count fingers at five feet, and read No. 14 of Jaeger's test-types. Left vision for distance, one fifth of the normal; for near objects, no worse than the right. Hearing normal; general health good. No history of syphilis. On the 14th day after his first visit right vision had become reduced to mere perception of light, and later was lost entirely. On the 32d day left vision had sunk to less than one twentieth of the normal. On the 37th day he could only count fingers at four feet, and before the end of the third month, like its fellow, was totally blind. Hearing then began to be affected, on which side first being undetermined. Before the end of the sixth month he was completely deaf. The sense of smell was likewise impaired, and aqua ammonia held beneath the patient's nose produced no irritating or disagreeable effect. Finally, taste was evidently lost. The sense of touch alone remained with which to communicate. Speech gradually became incoherent; he lost flesh and strength, and finally died after lingering nine months. On *post-mortem* examination a fibro-sarcoma was discovered, the size and shape of an eye, well defined and involving the inferior portion of the right anterior lobe; the underlying dura was adherent about the cribriform plate, and considerable hyperæmia existed as far back as the anterior part of the pons.

The writer states: "It is difficult to understand how this nerve (Auchtung) on both sides could have its function entirely destroyed, while the motor oculi patheticus and trigeminus, the abducens, the portio dura, and the glosso-pharyngeal were apparently in perfect condition. Not the least sign of paralysis about the muscles of the eye or elsewhere, nor any diminished acuteness of sensation could be perceived during life, and after death these nerves appeared also to be in a healthy condition."—*Buffalo Med. and Surg. Jour.*, Feb., 1882.

THE DANGERS OF NERVE-STRETCHING.—Under the above

title, Dr. Julius Althaus, in referring to the dangers of the operation of nerve-stretching in locomotor ataxia, says: "It may, therefore, not be out of place to mention that at least five fatal cases have already been recorded—one by Socin, another by Langenbuch, who originated the operation; a third by Billroth and Weiss, a fourth by Berger, and a fifth by Benedict. In most of these cases the cause of death appears to have been undue violence used in stretching, whereby the medulla oblongata would appear to have received a shock. Thus, in Benedict's case, severe vomiting and isingultus, together with complete paralysis of the bowels and bladder, supervened after the operation; dyspnœa and cyanosis eventually set in, and the patient died comatose on the ninth day.—*Brit. Med. Jour.*, Jan. 7, 1882, p. 11.

W. R. BIRDSALL, M.D.

---

f.—THERAPEUTICS OF THE NERVOUS SYSTEM.

ARTIFICIAL CURARE.—*Prog. Méd.*, March 4, 1882. M. Rabeteau has found a compound with physical, chemical, and physiological properties exactly like those of curare. The compound is called the iodide of méthyl triethyl stibonium, the formula of which is

$$\left.\begin{matrix} C\ H_3 \\ C_2H_5 \\ C_2H_5 \\ C_2H_5 \end{matrix}\right\} Sb\ I.$$

Among the physiological effects are none attributable to the antimony in the compound.

NERVE-STRETCHING.—*Prog. Médical*, March 11, 25, and April 15, 1882. Review by Duret and Bonnaire.

Idiopathic sciatica.—Eleven cases treated; nine successful.

Symptomatic sciatica (diseases of the spinal cord).—Eight cases treated. In one, apparent cure; in five, marked alleviation of symptoms; and in two no change resulted.

Trigeminal neuralgia.—Different branches were stretched in twelve cases. Complete relief occurred in nine; partial in one; and no relief in two cases.

Intercostal neuralgia.—One case treated, with no relief.